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Manipulation of End Tidal Carbon Dioxide Levels in Patients Undergoing Shoulder Surgery in

Beach Chair Position

by

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Manipulation of End Tidal Carbon Dioxide Levels in Patients Undergoing Shoulder Surgery in Beach Chair Position

Abstract

With the beach chair position (BCP) being used in the majority of surgical operations involving the shoulder, examination of harmful neurological sequelae is being brought under investigation into improving anesthetic technique and more specifically the ventilation strategies used to improve cerebral oxygenation. A number of complications have been identified including cranial nerve injury, visual loss, and cerebral infarction, which have occurred in individuals who have been considered relatively healthy patients (Moerman, De Hert, Jacobs, De Wilde & Wouters, 2012). In order to closely monitor these strategies, “near infra-red spectroscopy (NIRS) is being used as a non-invasive technology that allows real-time assessment of the adequacy of cerebral tissue oxygenation (and, indirectly, of CBF) in the operating theatre setting” (Murphy et al., 2014, p. 618).

Following induction of general anesthesia and successful intubation, common anesthesia technique involves hyperventilating the patient to achieve an end tidal carbon dioxide (EtCO₂) value of approximately 30 mm Hg. In patients undergoing surgery in the BCP, this strategy may in fact limit cerebral blood flow (CBF) by inducing cerebral vasoconstriction. Randomized studies conducted to evaluate the differences between two ventilation strategies on cerebral oxygenation demonstrated the positive effect of maintaining a higher EtCO₂ (Murphy et al., 2014; Picton et al., 2015).

This case study investigated on the pathophysiology and anesthetic management of an 89 year old, female patient, diagnosed with osteoarthritis of the shoulder who underwent a reverse total shoulder arthroplasty in the BCP under general anesthesia. This case study was compared

with current literature on appropriate anesthetic management of patients undergoing shoulder surgery in the BCP. Anesthetic goals for this particular patient population are preparation, maintenance of cerebral perfusion, prevention of hypotension and reduction of cerebral desaturation events (CDEs) throughout the perioperative course.

Keywords: beach chair position, ventilation, end tidal carbon dioxide, cerebral oxygenation

Background and Rationale

For patients undergoing surgical operation of the shoulder, the BCP is used 70% of the time for greatest surgical exposure and manipulation compared to the lateral decubitus position (Murphy et al., 2014). As an anesthesia provider, one of the most challenging aspects of this particular procedure is maintaining adequate cerebral perfusion pressures (CPP) in conjunction with cerebral oxygenation levels. If inadequate cerebral perfusion occurs for an extended period of time, increased risk for development of ischemic neurological injury becomes evident (Murphy et al., 2014). Although the risk of perioperative stroke is quite low at 0.08%-0.7% for patients undergoing a general operation (not vascular), an inherent risk is evident when the BCP is used in conjunction with general anesthesia (Selim, 2007).

In regards to strokes related to the BCP, limited data has been presented reflecting concrete evidence that indeed indicate strokes are due in part to the upright position. However, four case studies have reported ischemic brain injury and spinal cord injury after surgery in the BCP (Pohl & Cullen, 2005). Their stance on the reports “suggests that the sitting position and the head position create specific physiological conditions that may be conducive to cerebral and spinal cord ischemia during this type of surgery” (Pohl & Cullen, 2005, p. 463).

As a patient is moved from the supine position to the sitting position while under general anesthesia, mean arterial pressure (MAP) at the level of the Circle of Willis is reduced by approximately 2 mm Hg per inch (or 0.77 mm Hg per cm) above the heart or level of measurement (Nagelhout & Plaus, 2014). For an average adult, this can equate to a drop in MAP of 20-24 mm Hg at the level of the Circle of Willis, not to mention an even greater drop at the most cephalad portion for the brain. The body’s visceral organs thrive with a MAP greater than

65mm Hg, which requires anesthesia personnel to maintain a MAP via blood pressure (BP) readings of 85-90 mm Hg in order to maintain an adequate level of cerebral perfusion.

The obstacle anesthesia personnel encounter with maintaining this elevated level of BP for extended periods of time is surgical site blood loss. As many surgeons will attest, maintaining a BP as low as possible without affecting neurological status is in their best interest. This is where the role of carbon dioxide levels come into play with helping to uphold cerebral blood flow (CBF) and cerebral oxygenation while teetering the line of an adequate MAP. The three factors which play a key role in CBF include arterial carbon dioxide (PaCO_2), arterial oxygen (PaO_2) and temperature; of these 3, PaCO_2 levels is the most important (Nagelhout & Plaus, 2014). Hypercarbia results in increased CBF through cerebral vasodilation as compared to hypocarbia, which results in decreased CBF through cerebral vasoconstriction. Normal CBF is 50ml per 100g of brain tissue per minute (Butterworth, Mackey, & Wasnick, 2013). For each 1 mm Hg increase in PaCO_2 , CBF increases 1-2ml per 100g of brain tissue per minute (Butterworth, Mackey, & Wasnick, 2013).

“Several factors may contribute to reductions in CBF during BCP shoulder surgery, which include patient positioning (hypotension in the sitting position), use of anesthetic agents (venous pooling in the lower extremities, reductions in cardiac output), flexion or extension of the head (mechanical obstruction of arterial or venous vessels) or mechanical ventilation (hyperventilation and positive pressure ventilation)” (Murphy et al., 2014, p. 618). The use of cerebral near-infrared spectroscopy (NIRS) has been instituted for many of these procedures in the BCP in order to better evaluate cerebral oxygenation.

This project will investigate the effects of CO_2 levels as it relates to cerebral oxygenation/perfusion in a patient undergoing shoulder surgery in the BCP. Current literature

will be used to illustrate the added benefits of driving EtCO₂ values higher than traditionally practiced in order to provide improved cerebral blood flow which in turn decreases risk of potential life altering neurological sequelae from occurring postoperatively in the discussed patient population.

Case Report

An 89 year-old, 162cm, 79 kg patient with a history of osteoarthritis of the shoulder presented for right reverse total shoulder arthroplasty. The patient's history included the following: dyslipidemia, atrial fibrillation, hypertension, chronic kidney disease stage 3, cerebrovascular disease, transient ischemic attacks, gastroesophageal reflux disease, diabetes mellitus type 2, enchondroma and glaucoma. Prior surgical history included total knee arthroplasty, cholecystectomy, shoulder arthroscopy and appendectomy with no indications of prior anesthetic complications. Patient's allergies included hydrocodone, diltiazem and amoxicillin. Home medications included simvastatin, tramadol, senna, warfarin, metoprolol, aspirin, pantaprazole and gabapentin. Preoperative laboratory data included hemoglobin of 14.3 g/dl, hematocrit of 43.3%, sodium of 139 mEq/L, potassium of 4.6 mEq/L, creatinine of 1.23 mg/dl, glucose of 155 mg/dl, prothrombin time of 22.7 seconds and an international normalized ratio of 2.0. Preoperative airway assessment revealed a mallampati 2 classification with limited neck range of motion. Patient had upper and lower dentures which were removed prior to entering the operating room (OR). The patient was given an ASA classification of 4.

An interscalene block was performed by the anesthesiologist in the pre-op holding room. For the block, 50mcg fentanyl IV was given along with localization at the insertion site with 1% lidocaine. With use of the nerve stimulator, the brachial plexus was located. Injection around the

plexus was carried out with 32 ml of local anesthetic consisting of a 3:1 ratio 0.25% bupivacaine with epinephrine and 0.5% ropivacaine.

Upon entering the OR, standard monitors were applied along with cerebral oximetry probes which were applied bilaterally to the forehead prior to oxygenation. A baseline level was obtained and recorded as 68% on the left and 62% on the right. Pre-oxygenation was then initiated. An arterial line was placed via the right radial artery. The peripheral intravenous catheter on the ipsilateral arm was inadvertently removed. Decision was made to insert left internal jugular central venous catheter for vascular access.

Prior to induction, oxygen was administered by face mask with 10L/min of 1.0 fraction of inspired oxygen. Anesthesia induction agents were as follows: 100 mcg fentanyl, 50 mg lidocaine and 12 mg etomidate. Following confirmation of manual ventilation, 50mg rocuronium was administered. The trachea was intubated with a 7.0 endotracheal tube using a Mac 3 blade via one attempt. A grade 1 view was obtained and confirmation of tube placement via EtCO₂, bilateral breath sounds via auscultation, bilateral chest rise and condensation in endotracheal tube. The tube was secured via tape, eyes taped and general anesthesia was maintained with sevoflurane 1.3% inspired concentration in a mixture of oxygen 1L/min and air 1L/min. Mechanical ventilation was set to volume control with tidal volume of 500 ml, respiratory rate of 13 breaths per minute (bpm) and positive end expiratory pressure of 4 cmH₂O .

Following induction, patient was placed in BCP with head piece placed by the physician's assistant (PA) and confirmation of neutral head/neck alignment confirmed via anesthesia staff and PA. Soon after positioning into the BCP, the patient's systolic blood pressure (SBP) dropped from pre-op levels of approximately 150 mm Hg to approximately 100 mm Hg.

A matching decline in cerebral oxygenation occurred quite rapidly. A phenylephrine drip was initiated via micro drip tubing and set to gravity.

Initial EtCO₂ values were aimed at achieving a level of 30-32 mm Hg. Even though SBPs were brought back to pre-op levels, cerebral oximetry values remained 15-20% below baseline. Ventilator changes were made by decreasing respiratory rates from 13 to 6 bpm in order to achieve EtCO₂ values of 40-43 mm Hg. In doing so, cerebral oximetry values quickly rose to levels approaching and even exceeding baseline values.

Total intravenous fluids administered was 1500ml, blood loss of approximately 50ml and urine output of 400ml was observed. Post-operative nausea and vomiting prophylaxis was achieved using 4mg of ondansetron and 10mg of dexamethasone. Emergence was initiated upon surgical closure. Extubation was performed following patient being able to maintain tidal volumes, respiratory rate greater than 8, hemodynamically stable (phenylephrine discontinued) and able to follow commands. Patient was then transferred to the post anesthesia care unit with 2 liters oxygen via nasal cannula. Total anesthesia time for the case was 3 hours and 23 minutes.

The patient was transferred to the floor for observation. Patient was then discharged the following day to the nursing home for recovery prior to returning to home. No documentation of neurological deterioration was noted during hospitalization.

Discussion

Cerebral Blood Flow

Anteriorly, the brain's blood supply is via the two internal carotid arteries and posteriorly, via the vertebral arteries (Porth & Matfin, 2009). These two sets of arteries communicate with each other at the base of the brain by way of the circle of Willis; in doing so, if one of these main arteries becomes stenosed or blocked, adequate blood supply to the brain

will remain sufficient (Porth & Matfin, 2009). “Without collateral input, cessation of blood flow in cerebral arteries results in ischemic neural damage as metabolic needs of electrically active cells exceed nutrient supply” (Porth & Matfin, 2009, p. 1317). Beyond the circle of Willis, an intricate network of arteries comprise the brain’s blood supply. The venous system is made up of two sets of veins which empty into the dural sinus: the deep or great cerebral venous system and the superficial venous system (Porth & Matfin, 2009).

With the brain using 15% to 20% or approximately 750 ml/minute of the average adults cardiac output, coordinated regulation of cerebral blood flow is paramount in maintaining adequate oxygenation to the cerebral tissues. Cerebral blood flow in the average adult is approximately 50 ml/100 g/min (Nagelhout & Plaus, 2014). Flow rates must remain preferentially above 25 ml/100 g/min in order to preserve cerebral function (Butterworth, Mackey & Wasnick, 2013). Due in part to the high metabolic demands of neuronal tissue, a balanced control between neuronal activity and cerebral blood flow within the brain, known as functional hyperemia, is vital in maintaining cerebral homeostasis (Cipolla, 2009).

Two important laws help in determining and demonstrating cerebral blood flow: Ohm’s law and Poiseuille’s law. Ohm’s law states flow is equal to the change in pressure from one end of the vessel to the other divided by the resistance of the vessel to the flow (Nagelhout & Plaus, 2014). “Poiseuille’s law states flow is directly related to the change in pressure, blood viscosity and the length of the vessel and inversely related to radius to the fourth power” (Cipolla, 2009, p. 47). Furthermore, the radius is ultimately the most important factor involved in determining cerebral blood flow (Paulson, Strandgaard & Edvinsson, 1990). Carbon dioxide, one of the most potent factors in regulation of the radius of the cerebral vessels, will be discussed later in this review.

Measuring CBF

Measurement of cerebral blood flow can be accomplished via a number of methods. A transcranial Doppler can be used by way of an ultrasound probe being placed over the temporal area which captures the velocity of blood flow in the middle cerebral artery; normal velocity speed is approximately 55 cm/sec (Butterworth, Mackey & Wasnick, 2013). Another method used is measuring brain tissue oxygen tension “through the placement of a bolt with a Clark electrode oxygen sensor; normal brain tissue oxygen tensions varies from 20-50 mm Hg” (Butterworth, Mackey & Wasnick, 2013, p. 577). Intracranial microdialysis can be used which measures the differences in the chemistry of the brain tissue, examining for levels indicating ischemia or injury to the brain (Butterworth, Mackey & Wasnick, 2013). Finally, the use of near infrared spectroscopy can be used to measure the cerebral oxygenation. Any decline in cerebral oxygenation is indicative of cerebral desaturation or impaired oxygen delivery to the brain.

Regulation of Cerebral Blood Flow

Cerebral blood flow is a complex process involving a multitude of factors contributing to the regulation of adequate oxygen supply to the cerebral tissues. Regulation of cerebral blood flow is primarily dependent on the following: cerebral perfusion pressure, cerebral autoregulation and extrinsic factors such as respiratory gas tensions, temperature, viscosity of the blood and autonomic influences (Butterworth, Mackey & Wasnick, 2013). Cerebrovascular congenital anomalies have also been implicated in disrupting cerebral circulation in up to 50% of patients (Koh, Levin, Chehab & Murphy, 2013). In conjunction with these influences, patients undergoing an operative procedure have additional factors influencing cerebral blood flow which may include positioning, anesthetic agents administered and mechanical ventilation (Murphy et al., 2014).

Cerebral perfusion pressure. Cerebral perfusion pressure is calculated as the difference between the mean arterial pressure and intracranial pressure (ICP) or central venous pressure (CVP), whichever is greater (Butterworth, Mackey, & Wasnick, 2013). CPP goals are set at 80-100 mm Hg with the most critical determinant being MAP (Butterworth, Mackey, & Wasnick, 2013). Severe increases in ICP can have deleterious effects on CPP resulting in cerebral ischemia due in part to cerebral hypoperfusion (Dagal & Lam, 2009). The three factors comprising ICP are blood volume, cerebrospinal fluid (CSF) and brain matter. Any increase in these factors may compromise CPP. Electroencephalogram (EEG) readings will begin to show areas of slowing when CPP is less than 50 mm Hg, areas of flatness when CPP is less than 25-40 mm Hg and may result in irreversible damage when levels are less than 25 mm Hg for any period of time (Butterworth, Mackey, & Wasnick, 2013).

Cerebral autoregulation. There are a number of autoregulatory processes present in the human body but none more developed than in the brain which is to be expected due to the need for continual blood supply, metabolic demands and water homeostasis (Cipolla, 2009). The brain's ability to autoregulate cerebral blood flow is relatively constant despite continual changes in CPP. "Autoregulation is an active vascular response characterized by (1) arterial constriction when the blood pressure is increased and (2) arterial dilation in response to decreases in systemic blood pressure" (Hines & Marschall, 2012, p. 219). Cerebral autoregulation maintains cerebral blood flow when mean arterial pressures are maintained between 60 and 160 mm Hg (Nagelhout & Plaus, 2014). Although literature varies slightly on this range of MAP, each source is within 10 mm Hg for the upper and lower ranges listed above. Cerebral blood flow is relatively constant across this wide range of blood pressure which "requires reflex adjustments in cerebrovascular resistance concomitant with changes in blood pressure" (Willie, Tzeng, Fisher, & Ainslie, 2014,

p. 846). This process is what allows the cerebral cortex to adjust the cerebral blood flow in localized areas to meet the metabolic needs (Porth & Matfin, 2009).

When an individual's MAP is consistently outside of these ranges such as in chronic hypertensive patients, the range is then shifted. Hypertensive patients for example, are shifted to the right, thus requiring higher CPP and MAP in order to ensure adequate cerebral perfusion (Cullen & Kirby, 2007). In acute instances, the brain's ability to autoregulate becomes compromised, disrupting the blood-brain barrier and posing a risk for cerebral edema and hemorrhage (Butterworth, Mackey, & Wasnick, 2013). Outside of the normal autoregulation range, cerebral blood flow becomes linearly dependent upon MAP (Cipolla, 2009).

When hypoperfusion or hypotension occur and autoregulatory processes are abolished, cerebral ischemia ensues (Cipolla, 2009). For a brief period of time, this reduction in CBF is metabolically compensated for by increased oxygen removal from the red blood cells. Signs and symptoms of cerebral ischemia are not seen until the metabolic needs of oxygen are unmet; dizziness, altered mental status and infarction are the eventual consequences (Cipolla, 2009).

Myogenic. Cerebral autoregulation is influenced by two mechanisms: myogenic and metabolic. Myogenic mechanisms encompass an intrinsic arterial response to varying mean arterial pressures (Butterworth, Mackey, & Wasnick, 2013). Myogenic tone involves vasodilation and vasoconstriction in response to changing cerebral perfusion pressures. When blood pressures rise, the myogenic response results in vasoconstriction which thus limits increased blood flow. To the contrary, when blood pressure diminishes, vasodilation ensues supplying the brain with increased blood flow. When mean arterial pressures stray outside of the normal ranges, myogenic tone is compromised. For example, during acute hypertension, myogenic constriction is overcome by the extreme intravascular pressure and forcible dilation of

the vessels (Cipolla, 2009). When myogenic tone is overcome by increasing blood pressures, blood volume uninhibitedly rises. This process is known as autoregulatory breakthrough (Cipolla, 2009). Devastating consequences involved may include hypertensive encephalopathy, posterior reversible encephalopathy syndrome and eclampsia due in part to the increased hydrostatic pressure causing cerebral edema (Cipolla, 2009).

Metabolic. Metabolic mechanisms are also at work within the brain. These mechanisms have been indicated for the responsibility of arteriolar tone (Butterworth, Mackey, & Wasnick, 2013). For example, when cerebral tissue demands outweigh the blood flow, metabolites are released to cause vasodilation in order to increase blood flow; the hydrogen ion being one of these metabolites (Butterworth, Mackey, & Wasnick, 2013). Increased levels of hydrogen ions cause an increase in cerebral blood flow in a response to wash away the acidic materials per se (Porth & Matfin, 2009). Other vasoactive mediators found to have vasomotor effects on the cerebral vessels include, but are not limited to, nitric oxide, acetylcholine, Gamma-Aminobutyric acid (GABA) and serotonin.

Extrinsic factors.

PaCO₂. The effects of arterial oxygen and carbon dioxide have been discussed in the literature as having profound effects on cerebral blood flow. Of these two factors, arterial carbon dioxide (PaCO₂) has the most profound effect, causing potent vasodilation during periods of hypercapnia and vasoconstriction during periods of hypocapnia (Meng & Gelb, 2014). With that being said, homeostatic mechanisms are said to be kept intact when PaCO₂ tensions are kept between 20 and 80 mm Hg (Butterworth, Mackey, & Wasnick, 2013). Outside of this range, cerebral blood flow becomes pressure dependent. Cerebral blood flow changes 1-2ml/100 g/min per mm Hg change in PaCO₂. This effect is immediate and is thought to be secondary to the

changes it elicits on the pH of CSF and cerebral tissue (Butterworth, Mackey, & Wasnick, 2013). Decreases in CSF pH ultimately resulting in vasodilation and increases result in vasoconstriction (Hines & Marschall, 2012).

The blood-brain barrier plays an important role in this process as well. Ions do not readily cross the blood-brain barrier, thus, CO₂ has a much more profound effect than does bicarbonate and/or hydrogen ions (Butterworth, Mackey, & Wasnick, 2013). Following the acute phase of hypercapnia or hypocapnia, metabolic processes do eventually help to compensate through increasing or decreasing bicarbonate levels in the CSF, but this process can take up to 24-48 hours (Butterworth, Mackey, & Wasnick, 2013). Hines & Marschall (2012), argue “this adaptive change, which reflects active transport of bicarbonate ions into or from the CSF, requires approximately 6 hours to return the CSF pH to normal” (p. 219). Keep in mind the aforementioned range of 20-80 mm Hg being the range in which CBF is adequately regulated via autoregulation. “Anesthetic drugs, however, alter resting cerebral blood flow and therefore, can change the cerebral blood flow response to changes in PaCO₂” (Pohl & Cullen, 2005, p.467)

With regards to hypercapnia, Meng & Gelb (2014), postulated two important aspects to contemplate when facilitating hypercapnia under anesthesia: how it affects the lower and upper limits of cerebral autoregulation when combined with hypotension and hypertension respectively. “The combined vasodilatory effects imposed by hypotension and hypercapnia could shift the lower limit rightward. Likewise, the dilation induced by hypercapnia could adversely affect the hypertension-induced constriction, rendering a leftward shift of the upper limit” (Meng & Gelb, 2014, p. 197). Severe hypercapnia will result in cerebral vessels being maximally dilated and consequently the pressure-flow relationship in the brain will be linear (Meng & Gelb, 2014). In a study conducted by McCulloch, Visco, & Lam (2000), they indicated that the “threshold at

which hypercapnia significantly impaired autoregulation averaged 56 mm Hg during sevoflurane anesthesia and 61 mm Hg during propofol anesthesia” (p. 1206).

PaO₂. Arterial oxygen content can also play a role in CBF, but must be a marked change. Hypoxia in particular is associated with cerebral vasodilation. Hyperoxia is associated only with limited vascular constriction (Butterworth, Mackey, & Wasnick, 2013). Cerebral blood flow does not change drastically until levels of PaO₂ fall below 50 mm Hg (Cipolla, 2009). As hypoxia progressively worsens, cerebral blood flow can rise by an estimated 400% of resting levels (Cipolla, 2009). This direct effect of vasodilation is found in the vascular cells of cerebral arteries and arterioles (Cipolla, 2009). Hypoxia also induces other mechanisms of vasodilation including the increased production of adenosine, nitric oxide, cyclic nucleotides and adenosine triphosphate sensitive potassium channels (Meng & Gelb, 2014). With vasodilation being a direct effect of hypoxia, intentionally inducing hypoxia during an anesthetic to provide greater cerebral blood flow through vasodilation would not be advantageous, merely a physiologic response of inadequate oxygen supply.

Temperature. Temperature regulation of patients undergoing anesthesia is paramount to the practice of patient safety. Inadequate temperature regulation has such implications of decreased wound healing, increased oxygen consumption, increased risk of myocardial infarction and prolonged hospital stays (Nagelhout & Plaus, 2014). Cerebral blood flow is also greatly affected by temperature regulation. As the temperature is increased or decreased by 1 degree Celsius, blood flow changes 5% to 7% (Butterworth, Mackey, & Wasnick, 2013). Hypothermia decreases cerebral blood flow and autoregulation whereas hyperthermia does just the opposite (Dagal & Lam, 2009).

Viscosity. The viscosity of blood can greatly affect the rate and turbulence of cerebral blood flow. The single most important determinant of viscosity is hematocrit (Pohl & Cullen, 2005). When reviewing laboratory data, an elevated hematocrit can inhibit blood flow and to the contrary, a low hematocrit can greatly increase cerebral blood flow. While hemodilution reduces viscosity and increases blood flow, it also decreases vascular tone which in turn limits autoregulation (Dagal & Lam, 2009). The drawback to a decreased hematocrit is limited oxygen carrying capacity of the blood with an optimal level of hematocrit being estimated at 30% (Butterworth, Mackey, & Wasnick, 2013).

Autonomic influences. With the deep cerebral blood vessels being primarily controlled via autoregulation, the superficial and major blood vessels of the brain are innervated via the autonomic nervous system (Porth & Matfin, 2009). More specifically, the sympathetic nervous system acts upon the large and intermediate sized blood vessels during times of stress when local mechanisms fail. For example, when hypertension occurs during activity, strain, pathology and during rapid-eye movement sleep, the sympathetic nervous steps in to vasoconstrict the large and intermediate vessels in order to preserve the smaller vessels from exhaustive pressures (Willie et al., 2014). These same protective reflexes may be responsible for vasospasms following stroke, aneurysm or brain injury (Butterworth, Mackey & Wasnick, 2013). Under anesthesia, sympatholytic drugs are used which indirectly exert their effects on inhibiting these sympathetic responses. Cerebral blood flow then becomes more pressure passive when sympatholysis has occurred (Willie et al., 2014).

Effect of Anesthetic Agents on Cerebral Blood Flow

Volatile anesthetics. All volatile anesthetics produce a global cerebrovascular dilating effect which results in increased blood flow, increased ICP and increased blood volume. Volatile anesthetics also cause a decrease in cerebral metabolic rate of oxygen consumption. An impairment of cerebral autoregulation occurs on a dose dependent manner (Butterworth, Mackey, & Wasnick, 2013). With the decrease in systemic vascular resistance imposed by volatile anesthetics, mean arterial pressures are decreased causing a reflective decrease in cerebral perfusion pressures (Nagelhout & Plaus, 2014). “Proposed mechanisms related to the neuroprotective effect of inhalation anesthetic agents include activation of ATP-dependent potassium channels, up-regulation of nitric oxide synthase, reduction of excitotoxic stressors and cerebral metabolic rate, augmentation of peri-ischemic cerebral blood flow and up-regulation of antiapoptotic factors” (Nagelhout & Plaus, 2014, p. 700-701).

As discussed earlier, carbon dioxide has potent effects on cerebral blood flow. When efforts are made to hyperventilate or hypoventilate, the response to the cerebral vasculature is unchanged with volatile anesthetics with the exception being Halothane (Butterworth, Mackey & Wasnick, 2013). For example, when hyperventilation is attempted to produce hypocapnia (which causes vasoconstriction), the initial effects of the volatile agents are blunted. “Differences exist among the volatile agents in their ability to interfere with the cerebral vasculature’s responsiveness to CO₂. Variables that affect the reported differences include the type of surgical procedure the patient is undergoing, associated pathophysiology and the presence of any coexisting disease(s)” (Nagelhout & Plaus, 2014, p. 89).

Propofol. Propofol has indirect effects on reduction of cerebral perfusion pressures via dose dependent reductions in systemic blood pressure. Reductions in cerebral blood flow may be as much as 40% to 50% and are dependent on systemic reductions in blood pressure (Nagelhout

& Plaus, 2014). Decreased cerebral blood flow rates following propofol administration is prospectively due to decreased metabolic demands and cerebral vasoconstriction (Nagelhout & Plaus, 2014). In terms of autoregulation and reactivity to CO₂ changes, propofol has been shown to have little to no effect (Dagal & Lam, 2009).

Etomidate. Cerebral blood flow is reduced with the induction agent etomidate. With the hemodynamic stability of etomidate, cerebral perfusion pressures are maintained (Nagelhout & Plaus, 2014). Use of etomidate is limited due to its potentiality to cause myoclonic movements, post-operative nausea and vomiting and adrenal insufficiency.

Benzodiazepines. Benzodiazepines cause a dose dependent decrease in cerebral blood flow and have limited effects on cerebral perfusion pressures except in the case of elderly and unstable patients (Butterworth, Mackey & Wasnick, 2013).

Opioids. In general, opioids have limited effect on cerebral blood flow and cerebral perfusion pressures. Secondary causes to changes in cerebral blood flow are the result of hypoventilation causing a rise in PaCO₂ (Butterworth, Mackey & Wasnick, 2013). Likewise, changes in cerebral perfusion pressures are observed when systemic blood pressures are compromised following opioid administration (Butterworth, Mackey & Wasnick, 2013).

Ketamine. Ketamine produces a profound vasodilatory effect on the cerebral vasculature resulting in a 50% to 60% increase in cerebral blood flow (Butterworth, Mackey & Wasnick, 2013). This is the only intravenous anesthetic agent which causes increased cerebral blood flow. Cerebral vasculature response to carbon dioxide remains intact with use of ketamine (Nagelhout & Plaus, 2014).

Neuromuscular Blockers. Non-depolarizing agents appear to have negligible effects on cerebral blood flow unless a histamine response results in reduction of systemic blood pressures

(Butterworth, Mackey & Wasnick, 2013). The depolarizing agent, succinylcholine, has been shown to cause transient elevations in cerebral blood flow as a result of myotonic type movements. These movements can be attenuated by prior administration of non-depolarizing agent (de-fasciculating dose) and lidocaine (Nagelhout & Plaus, 2014).

Vasopressors. In patients with an intact cerebral autoregulatory system, vasopressor agents such as norepinephrine and phenylephrine, do not impact cerebral blood flow unless the systemic blood pressure is outside of the autoregulatory range (Butterworth, Mackey & Wasnick, 2013). When cerebral autoregulation is disrupted, cerebral blood flow is increased in a dose dependent manner.

Vasodilators. Cerebral blood flow is primarily increased in a dose dependent manner via vasodilating agents (Butterworth, Mackey & Wasnick, 2013). Sodium nitroprusside and nitroglycerin are smooth muscle relaxants which have direct cerebrovascular dilating effects which in turn increase cerebral blood flow and cerebral blood volume (Nagelhout & Plaus, 2014).

Beach Chair Positioning

The beach chair position (or sometimes referred to as the lounging, barbershop, or lawnchair position) has been used since the 1980's primarily by orthopedic surgeons who are attempting to gain greater visualization and mobilization of the shoulder and potentially decreasing risk of brachial plexus strain, a decreased risk of direct neurovascular trauma as compared with lateral decubitus position and ease of conversion to open approach (Murphy et al., 2010). Variations of this position range from the patient being at 45 degree to 90 degree angle in the sitting position. The patient's head is securely strapped in a horseshoe headrest with straps going circumferentially around the head in both the coronal and transverse planes in order

to provide a stationary position. The non-operative arm is placed on an armrest. Pillows or other padding devices are placed under the knees of the patient along with padding of the heels.

Careful observation of patient alignment and padding of pressure points prior to surgical draping is of supreme importance in order to prevent potential positioning injuries. Surgical manipulation of the arm can be forceful which in turn poses risk of re-positioning of the patient into an unfavorable position. Although adverse consequences have rarely been documented in patients in the BCP, potentially devastating neurological sequelae can occur resulting in stroke and spinal cord injuries (Kohet al., 2013).

Preventing flexion or extension of the head and neck is vitally important in order to maintain normal blood flow and venous drainage from the brain. “Blood flow reduction in the vertebral artery caused by extension and rotation or tilt of the head may result in posterior brain circulation infarcts” (Cullen & Kirby, 2007, p. 27). Endotracheal tube securement also needs to be judiciously observed as repositioning from the supine position to the sitting position as well as the erratic manipulations by the surgeon during the procedure places great risk on inadvertent extubation.

Normal physiologic changes in a non-anesthetized patient put into the BCP is compensation of hemodynamic changes via increased systemic vascular resistance by up to 50% to 80% (Pohl & Cullen, 2005). In anesthetized patients however, their ability to compensate for decreased mean arterial pressures and cardiac output are abolished by way of autonomic responses being blocked from anesthetic agents (Cullen & Kirby, 2007). Venous pooling ensues while under general anesthesia and is worsened in the upright position. Cerebral perfusion pressures are diminished secondary to the fall in mean arterial pressures in the anesthetized patient and are further worsened in the BCP. Remembering cerebral perfusion pressures maintain

autoregulatory function until hypotension (or hypertension) occurs; when hypotension occurs outside of the autoregulatory range, cerebral perfusion becomes completely dependent upon MAP.

Normally, cerebrovascular venous return is increased in the BCP, but under general anesthesia, the use of mechanical ventilation imposes positive pressure which acts as an obstruction to the superior vena cava decreasing venous return. Careful consideration to the level of positive end expiratory pressure (PEEP) being used should be considered. One advantage to the BCP in terms of ventilation is it allows a more normal physiologic position aiding in decreasing ventilation/perfusion mismatching (Nagelhout & Plaus, 2014).

In shoulder procedures, general anesthesia is often accompanied with an interscalene brachial plexus block. This regional block provides ample surgical anesthesia along with excellent post-op pain control. The issue lies in the sympatholysis of surgical stimuli, further enhancing hypotensive and hypoperfusion episodes while in the BCP under general anesthesia. Also, hypotensive bradycardic episodes (HBEs) have been commonly reported when used alone or in combination with general anesthesia; HBEs are characterized as a “decrease in heart rate at least 30 beats/min within a 5 minute interval, any heart rate less than 50 beats/min and/or a decrease in systolic blood pressure below 90 mm Hg” (Nagelhout & Plaus, 2014, p. 1006). This mechanism is most notably the result of or worsened by the Bezold-Jarisch reflex (Soeding et al., 2011).

When moving a patient from the supine to the sitting position, consideration of the level of blood pressure measurement is key. As mentioned earlier, MAP decreases 2 mm Hg for every inch the circle of Willis is above the heart. This can easily equate to a 25 mm Hg difference if the patient is situated high in the BCP. The gradient will be even more drastic if a blood pressure

cuff is to be placed on the lower extremity. In an ideal situation, the blood pressure should be monitored at the level of the external auditory meatus (Cullen & Kirby, 2007). Unfortunately, most patients undergoing shoulder arthroscopy or arthroplasty do not have an intra-arterial catheter placed due to low risk of surgical complications of surgery and increased risks associated with invasive monitoring.

Efforts to maintain an adequate MAP in order to preserve adequate CPPs can be made by aggressive fluid administration to erase deficits, decreasing venous pooling via compression stockings and vasopressors to restore blood pressures to adequate levels (Nagelhout & Plaus, 2014). Ventilation strategies have recently been proposed to utilize the vasodilating effects of carbon dioxide on the cerebral vasculature in order to improve cerebral blood flow.

Use of Cerebral Oximetry

The use of cerebral oximetry or near-infrared spectroscopy, has slowly been integrating its way into anesthesia practices across the nation. Despite its simplicity and added monitoring profile, NIRS has yet to be adopted as a standard of care monitoring device. Primary uses of this non-invasive technology have been put to use in the areas of vascular and orthopedic surgeries with investigation into its use in thoracic, hepatobiliary, robotic-assisted and pediatric surgeries. With the concern of neurocognitive sequelae in patients undergoing shoulder surgery in the BCP, the use of cerebral oximetry adds an additional monitor in which to help detect early cerebral deoxygenation states. Early detection and prevention of cerebral desaturation events for any extended period of time is critical in proactively decreasing the risk of ischemic brain and spinal cord injury and/or infarction (Murphy et al., 2010).

This advancing technology captures continuous monitoring of cerebral oxygen saturation in the frontal cortex (Moerman & De Hert, 2015). NIRS allows for a non-invasive measuring of

relative concentrations of oxyhemoglobin and deoxyhemoglobin (Picton, Shanks, Dorje & Mashour, 2010). “Cerebral oximeters use the relative transparency of the skull to light and hemoglobin oxygenation in the cerebral cortex which can then be determined by a differential analysis of the light absorption between near and far-field photodetectors” (Koh et al., 2013, p.1325). An estimate of the balance between cerebral oxygen supply and demand is ultimately portrayed by NIRS (Picton et al., 2010). NIRS has been demonstrated in various studies to indicate early warning signs of cerebral hypoperfusion. The ideation of ventilation strategies to improve cerebral oxygenation in the anesthetic management of patients in the BCP, as estimated by cerebral oximetry, is slowly captivating anesthesia professionals across the nation.

CO₂ Management in the BCP

With an 80% cerebral desaturation rate occurring in patients monitored with NIRS while in the BCP under general anesthesia and controlled ventilation, greater efforts to manipulate ventilation strategies have been recently reviewed and implemented (Murphy et al., 2014). With the aid of cerebral oximetry, these ventilation strategies are taking shape. “Studies in supine awake volunteers and surgical patients have demonstrated that changes in ventilation and end-tidal carbon dioxide tension result in significant alterations in regional cerebral oximetry values” (Murphy et al., 2014, p. 619).

In a recent study performed by Murphy et al. (2014), regarding the use of ventilation strategies to improve cerebral oxygenation, two separate groups underwent shoulder surgery in the BCP. Both groups were managed identically with the only difference being the ventilation strategies used. The control group was managed with an EtCO₂ of 30-32 mm Hg in comparison to the study group which was managed with an EtCO₂ of 40-42 mm Hg. Incidences of cerebral desaturation events (CDEs) were 8% in the study group as compared to 56% in the control

group. No patients were reported to suffer from any neurological sequelae, but the authors did point out that “cerebral desaturations of 15%-25% have been associated with fainting, symptoms of cerebral ischemia in carotid endarterectomy patients, cognitive dysfunction after hip and cardiac surgery and longer PACU and hospital administrations after abdominal surgery” (Murphy et al., 2014, p. 624).

In a similar prospective interventional within-group study performed by Picton et al. (2015), two different anesthetics (desflurane vs total intravenous propofol) were incorporated in the study design looking at the changes in regional cerebral oximetry when ventilation adjustments were made throughout the procedure. Adjustments to FiO₂ and minute ventilation were made at five different set points. Results showed no significant interaction between the two anesthetic choices on cerebral oximetry. However, there was a significant difference in cerebral oximetry values when patient’s ventilation was changed from an FiO₂ of 0.3 and an EtCO₂ of 30 mm Hg to an FiO₂ of 1.0 and an EtCO₂ of 45 mm Hg. Data found a 14% increase in cerebral oximetry between the two set points. Patients in the BCP with the elevated EtCO₂ and increased FiO₂ actually were found to have higher cerebral oximetry values compared to the initial supine set point.

Similarly, Picton et al. (2010), postulated that the effects of increasing EtCO₂ (40-45 mm Hg) along with 1.0 FiO₂ in comparison to the use of an EtCO₂ value of 30-35 mm Hg with 0.3 FiO₂ would result in a greater cerebral oxygenation values in patients without vascular disease. Results showed a 7-8% improvement in regional cerebral oxygenation values when using NIRS monitoring. The authors concluded that quantifiable changes in cerebral oximetry could be obtained in anesthetized patients with the use of changing ventilation strategies (increasing FiO₂ and EtCO₂).

A similar prospective controlled study performed by Picton et al. (2010), was performed to determine if there was a difference in increasing FiO_2 or changing EtCO_2 in patients undergoing a carotid endarterectomy during carotid cross-clamping to evaluate if improved cerebral oxygenation values would indeed occur. Results of the study revealed a 6% increase in cerebral oximetry in unshunted patients and 3% increase in shunted patients when ventilation strategies were aimed at keeping EtCO_2 40-45 mm Hg as compared to an EtCO_2 value of 30-35 mm Hg. In addition to the elevated EtCO_2 values, 1.0 FiO_2 was administered. The authors concluded increasing FiO_2 values along with increasing EtCO_2 reliably shows improved cerebral oximetry values which in turn represent improved cerebral oxygenation.

In another observational study regarding the prevalence of cerebral oxygen desaturation in the beach chair position, Moerman et al. (2012), pointed out the positive correlations between improved regional cerebral oximetry, when mean arterial blood pressures and EtCO_2 are elevated. The authors went on to make the recommendation to avoid hypotension and low EtCO_2 .

Case Study

The patient in this case study did present with potential risk factors for decreased cerebral perfusion which included advanced age, dyslipidemia, atrial fibrillation, hypertension, chronic kidney disease stage 3, cerebrovascular disease, transient ischemic attacks, diabetes mellitus type 2 and surgical procedure being performed in the BCP. Following induction of anesthesia and repositioning into the upright position, MAPs, as well as cerebral desaturation to sub 20% baseline values ensued. Despite correction of blood pressure via phenylephrine to pre-operative levels, cerebral desaturation continued. Ventilatory efforts were then manipulated in order to achieve an EtCO_2 value of approximately 40 mm Hg. Within minutes, cerebral oxygenation

values rose to within baseline levels and even exceeded baseline levels towards the latter part of procedure.

Despite a lack of evidence of any neurological sequelae occurring postoperatively in the case report presented (due in part to the cerebral desaturation event which occurred), future management of this patient population would benefit from two strategies: 1) a slower induction in order to prevent a precipitous drop in hemodynamic status and 2) initial ventilation strategies aimed at achieving an EtCO₂ value of approximately 40 mm Hg.

Deleterious Effects of Increased EtCO₂ Values

With the intended nature to increase arterial carbon dioxide levels in the patient population discussed, one must address the potential complication of acidosis. As PaCO₂ increases, physiologic pH reciprocally declines; these values directly correlate the hypoventilatory state proposed. With carbon dioxide being 20 times more soluble than oxygen, signs of hypoxemia during states of acute hypercapnia occur prior to respiratory acidosis (Porth & Matfin, 2012). Under the controlled operative setting, adequate oxygen flows combat with these high levels of carbon dioxide. Hypercapnia without hypoxia is usually only observed in situations such as hypoventilation (Porth & Matfin, 2012).

A number of bodily functions are disrupted from states of hypercapnia including acid-base balance and kidney, nervous system and cardiovascular function (Porth & Matfin, 2012). Under normal bodily functions, increases in serum bicarbonate via renal reabsorption work to increase pH. This process makes only transient changes in the first 6 hours and does not make any substantial changes in pH correction until the 24-48 hour mark. As long as pH remains within normal limits, the most common complications of hypercapnia revolve around hypoxia, which normally accompanies hypercapnia. Fortunately for anesthesia providers, unless a

pathology exists (ventilation-perfusion mismatch), hypoxia should not be a factor if proper endotracheal tube placement is confirmed.

Another drawback to inducing a hypercapnic state, which may be an anesthesia provider's greatest hurdle, is stimulating an anesthetized patient to breath. "The arterial and cerebrospinal fluid partial pressures of CO₂ are probably the most important inputs to the brainstem centers for establishing the ventilatory rate and tidal volume; increases in PaCO₂ and decreases in PaO₂ induce stimulation of arterial or peripheral chemoreceptors, with the carotid bodies apparently exerting a much greater influence on the medullary respiratory centers than the aortic bodies" (Nagelhout & Plaus, 2014, p. 608-609). These peripheral chemoreceptors are said to play a much greater role than central chemoreceptors in the short-term responses to CO₂ (Nagelhout & Plaus, 2014).

Recommendations

Following the review of literature, a number of recommendations can be made. First of all, a number of authors have indicated increasing EtCO₂ values in patients undergoing surgery in the BCP, may indeed benefit from increased cerebral blood flow secondarily to increased CO₂ levels. Such values indicated are between 40-45 mm Hg. Also, the use of 1.0 FiO₂ has been indicated as an important adjunct to increased EtCO₂ values.

Secondly, vigilance by the anesthesia provider to maintain an adequate MAP is vitally important. Without an adequate MAP at the circle of Willis, cerebral blood flow will be compromised and thus negating any effort to improve cerebral oxygenation through improved cerebral blood flow. Careful consideration of the level of measurement must be taken into account when monitoring and manipulating MAP.

Thirdly, either the use of or advocating for the use of cerebral oximetry will help to improve patient safety and potentially decrease the risk of neurological sequelae from occurring in such cases. Cost can become an issue for some institutions which is why the widespread use of this monitoring tool has yet to be set as a standard of care. One cannot argue with the fact of using such a device in at risk patients undergoing surgeries in the BCP when time and time again studies have shown frequent cerebral desaturation rates when this position is used.

Lastly, making sure to double check head/neck alignment prior to draping in order to decrease risk of either arterial or venous obstruction if the neck were to be in a position other than anatomical midline. Doing so will help to prevent further cerebral blood flow compromise and ultimately decrease risk of neurological deficits from occurring.

Conclusion

Use of ventilation strategies in order to improve cerebral oxygenation in patients undergoing surgery in the BCP has not been a mainstay of anesthetic management and up until the last 5-6 years has only been marginally investigated. Two randomized controlled trials completed just recently have indicated that improved cerebral oxygenation values via NIRS monitoring have resulted from ventilation strategies aimed at increasing EtCO₂ in conjunction with increased FiO₂ (Murphy et al., 2014; Picton et al., 2015).

The anesthesia provider must have a thorough understanding of the anatomy and physiology of cerebral blood flow and the factors which contribute to increasing and decreasing; more specifically, the effects of carbon dioxide levels imposing an immediate vasodilatory action when levels are increased. Balancing hemodynamic management along with ventilation strategies in this particular patient population may indeed provide improved outcomes and at the very least, decrease risk of potential life altering neurological deficiencies. Also, maintaining

proper body alignment and more specifically, attaining neutral head and neck position in a manner which allows appropriate arterial blood flow and venous drainage, will aid in overall management of patients in the BCP. Though induced mild acidosis with increasing EtCO₂ is generally short lived in the operative arena, the risk of acidotic states intraoperatively needs to be carefully thought out and treated accordingly.

There remains debate into the management of such patients accordingly and until future large-scale studies are completed, the debate will remain whether or not to increase EtCO₂ values in order to improve cerebral oxygenation secondary to increased cerebral blood flow. Further studies are needed to determine if there is a cause and effect relationship between higher EtCO₂ values and decreased cerebral desaturation events leading to neurological deficits. In addition, further studies are needed in determining if indeed solely elevating EtCO₂ without increasing FiO₂ to 1.0 will illustrate similar results. Isolation of these two factors individually will help shed light into further support of the ventilation strategies proposed.

A case-by-case analysis along with a risk versus benefit approach should be reviewed by the practitioner prior to implementing such strategies. Consideration of pathologies and the surgical procedure for each patient is vitally important in providing safe and effective anesthesia.

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Manipulation of End Tidal Carbon Dioxide Levels in Patients Undergoing Shoulder Surgery in the Beach Chair Position

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Beach Chair Position

- Beach Chair Position (BCP) is the primary position for shoulder surgery
- Risk of perioperative cranial nerve injury, visual loss, and cerebral infarction increase with the inherent decrease in cerebral perfusion pressures when BCP is utilized
- Review of literature was conducted to examine clinical recommendations for the use of ventilation strategies in this patient population

Does increasing EtCO₂ in order to improve cerebral blood flow decrease risk of neurological sequelae from occurring?

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Cerebral Perfusion

- Brain uses 15-20% of Cardiac Output
- Under General Anesthesia, MAP at the Circle of Willis is reduced by approximately 2 mm Hg per inch above the heart **OR level of measurement**
- Typically a 20-24 mm Hg difference

Target MAP shifted from 65 to 85 mm Hg

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Intraoperative Strokes

- Risk of perioperative stroke 0.08-0.7% for general operations (non vascular)
- Limited data directly correlating strokes to BCP
 - Under reporting
- "The sitting position and the head position create specific physiological conditions that may be conducive to cerebral and spinal cord ischemia during this type of surgery" (Pohl & Cullen, 2005, p. 463)

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Cerebral Blood Flow

Regulation of CBF

- Cerebral perfusion pressures
 - MAP-ICP (or CVP whichever is greater)
 - 80-100 mm Hg
- Cerebral autoregulation
 - 60-160 mm Hg
- Extrinsic Factors
 - Respiratory gas tensions
 - Temperature
 - Blood viscosity
 - Autonomic influences

Factors Affecting CBF

- Patient position
- Anesthetic agents
- Flexion or extension of head/neck
- Mechanical ventilation

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Carbon Dioxide

- "Profound vasodilator"
- Homeostatic mechanisms kept intact with PaCO₂ tensions 20-80 mm Hg
 - Outside this range blood flow becomes pressure dependent
- Cerebral blood flow changes 1-2ml/100 g/min per mm Hg change in PaCO₂
- Crosses blood brain barrier=>immediate effect

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Case Study Information

- Right Reverse Total Shoulder Arthroplasty
- 89 year old Female
- 79 kg
- ASA 4
- Allergies: hydrocodone, amoxicillin, diltiazem

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Pre-operative Evaluation

- PMH: Dyslipidemia, atrial fibrillation, HTN, CKD, cerebrovascular disease, TIAs, GERD, DM type 2, enchondroma, and glaucoma
- Surgical Hx: TKA, cholecystectomy, shoulder arthroscopy, and appendectomy
- Medications: Simvastatin, tramadol, senna, warfarin, metoprolol, aspirin, pantoprazole and gabapentin
- Pre-op vitals: 113/87, HR 89, RR 16, T 36.7, SaO₂ 94 on RA
- Airway evaluation: MP 2, TM distance >3 FB, limited mouth opening, limited neck ROM and dentures

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Pre-operative Evaluation

Laboratory Data

- Hgb- 14.3
- Hct- 43.3
- Pits- 166
- Cr- 1.23
- K- 4.6
- INR- 2.0
- PT- 22.7

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Anesthetic Course

- GETA
 - Pre-oxygenated 10L via facemask
 - 100mcg Fentanyl
 - 50mg Lidocaine
 - 12mg Etomidate
 - + mask ventilation
 - 50mg Rocuronium
 - 7.0 ETT via Mac 3
 - +EtCO₂ and BIS
 - Sevoflurane 1.1-1.7%
- Interscalene Block
 - 50mcg Fentanyl
 - 32ml 3:1 ratio 0.25% bupivacaine with epi and 0.5% ropivacaine
- Additional Medications
 - Ondansetron 4mg
 - Decadron 10mg
 - Phenylephrine gtt
 - No additional paralytics or narcotics given

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Anesthetic Course

- Left radial arterial line placed
- Left IJ CVC placed due to marginal IV access and difficult venipuncture
- Cerebral Oximetry
- Standard Monitors

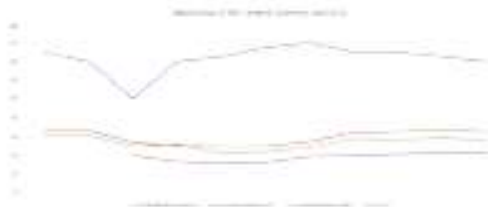
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Intraoperative Issues

- Hypotension gradually ensued following induction
 - Neo gtt initiated
- Cerebral oximetry values remained low despite return to pre-induction BPs
- Ventilation changes made to increase EtCO₂
- Cerebral oxygenation values quickly rose to baseline

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Intraoperative Issues



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PACU

- Uneventful emergence and PACU stay
 - Vital signs: BP 128/65, HR 78, RR 10, T 97.3, SaO2 97%
- Pt denied any pain postop
- No evident signs of neurological delays
- Case totals
 - LR 1500
 - Urine 575
 - EBL 50
 - Anesthesia time: 3 hours 58 minutes

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Discussion

Beach Chair Positioning

- Used for greater visualization, decreased neurovascular injury, and ease to open approach
- Autonomic reflexes abolished by GA
- Venous pooling ensues
- CPP decreased proportioning to decrease in MAP

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Regional + General

Interscalene Block (ISB)

- ISB typically used
- Sympatholysis further enhances hypotension/hypoperfusion

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Use of Cerebral Oximetry

- Near-Infrared Spectroscopy (NIRS)
- Primary use in vascular & cardiac arena
- Sparsely used as a standard monitoring tool
 - Used for patients at risk of cerebral desaturation events at facility
- Offers early detection and prevention of cerebral desaturation

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CO2 Management in the BCP

- According to Murphy et al. (2014), an 80% cerebral desaturation rate occurs in pts monitored via NIRS while in BCP
- "Studies in supine awake volunteers and surgical patients have demonstrated that changes in ventilation and EtCO2 tension result in significant alterations in regional cerebral oximetry values" (Murphy et al., 2014, p. 619)

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CO2 Management in the BCP

- Randomized controlled trial by Murphy et al. (2014) included 70 patients undergoing shoulder surgery in BCP
 - Control group=> EtCO2 30-32 mm Hg
 - Study group=> EtCO2 40-42 mm Hg
 - Cerebral desaturation events (CDEs) were 8% in study group compared to 56% in the control group

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CO2 Management in the BCP

- Prospective interventional within-group study by Picton et al. (2015) included 56 patients undergoing surgery in the BCP
 - Two anesthetics used (Desflurane vs. TIVA)
 - FIO2 and target EtCO2 adjustments made at 5 set points
 - No significant interactions between the two anesthetics
 - Between the two set points of FIO2 0.3/EtCO2 30 mm Hg and FIO2 1.0/EtCO2 45 mm Hg showed a 14% improvement in cerebral oxygenation

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CO2 Management in the BCP

- Prospective controlled study by Picton et al. (2010) included 20 patients undergoing carotid endarterectomy under GA
 - Study revealed 6% increase in cerebral oximetry in the unshunted patients and 3% increase in shunted patients when EtCO2 was kept at 40-45 mm Hg versus 30-35 mm Hg
 - FIO2 was kept at 1.0

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CO2 Management in the BCP

- Prospective pilot study by Picton et al. (2010) included 10 patients without vascular disease undergoing surgery under GA
 - Following induction and intubation, FIO2 and minute ventilation adjusted at set points
 - Results showed a 7-8% improvement in regional cerebral oxygenation from set point A (EtCO2 30-35 mm Hg/FIO2 0.3) to set point B (EtCO2 40-45 mm Hg/FIO2 1.0)

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Deleterious Effects of Increased EtCO2 Values

- Acidosis
 - Typically low end of normal pH
 - Brief
- Bodily functions disrupted from states of hypercapnia
 - Acid-base balance
 - Kidney, nervous system and cardiovascular function
- Stimulus for patient to breath

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Recommendations

- Aim to achieve EtCO2 values of approximately **40 mm Hg**
 - Use of 1.0 FIO2 also recommended per literature
- Use (or advocate for use of) cerebral oximetry to help closely monitor anesthetic course for pts in BCP
- Maintain adequate MAP
 - Remembering to compensate for level of BP cuff or transducer
- Aggressive fluid resuscitation to replace deficits
- Double check head/neck alignment

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Conclusion

- CO₂ potent vasodilator
- Induced mild acidosis short lived
- Balancing hemodynamic status with ventilation strategy
- Risk vs. Benefit
- Additional large-scale prospective studies needed

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